THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM MDMA

Using the Recovery Kernel Framework

MDMA · Ecstasy · Molly · E · X · Mandy · Pills · MDA · MDEA

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

MDMA HYPERTHERMIA IS THE PRIMARY ACUTE KILL MECHANISM.

If someone is very hot, confused, or stops sweating at a festival or event — call 911 immediately.

A Note Before You Begin

The compendium says it in a single line, and the line is exact: MDMA provides connection, love, and openness. The root deficit it addresses is the serotonin system and the social bonding system. The recovery architecture is authentic connection practices and community.

This is the only guide in this series where the primary function the substance provided was not stimulation, not calm, not escape, not pain relief, not performance enhancement — but love. The felt sense of genuine, open-hearted, barrier-dissolving love for the people around you. The removal of the social self-consciousness that normally mediates every human encounter. The direct, unmediated experience of connection.

Understanding this changes everything about the recovery framework. MDMA is not primarily a dopamine drug, though dopamine is involved. It is not primarily a GABA drug, though GABA is touched. It is primarily a serotonin drug — and specifically, a drug that floods the serotonin system, releases oxytocin (the bonding molecule), and temporarily removes the neurological barriers between the self and other people. The connection felt on MDMA is not an illusion — it is a real neurochemical state. It is also borrowed rather than built, which is the source of the problem.

MDMA recovery requires rebuilding the capacity for genuine connection through means the nervous system actually owns. That is both the challenge and the invitation.

A note on therapeutic MDMA: as of 2026, MDMA-assisted psychotherapy for PTSD is approved in Australia and has received FDA Breakthrough Therapy designation in the United States, with clinical availability expanding. This guide addresses MDMA use disorder — problematic patterns of recreational use. It does not address properly supervised therapeutic MDMA. If you are involved in legitimate clinical MDMA therapy, the relevant protocols and the therapeutic context are different from what this guide covers. The question 'can therapeutic MDMA be used in MDMA recovery?' is addressed in Section 8.

Section 0 — Immediate Safety: Read This First

⚠ MDMA HYPERTHERMIA AND HYPONATREMIA ARE THE TWO MOST COMMON CAUSES OF MDMA-RELATED DEATH. BOTH ARE PREVENTABLE AND BOTH ARE MEDICAL EMERGENCIES.

The Two Acute Kill Mechanisms

Emergency	What it is	Recognition and response
Hyperthermia (overheating)	MDMA raises core body temperature through serotonin-mediated mechanisms — affecting the hypothalamic thermostat. Physical exertion (dancing) and hot environments compound this dramatically. The body can reach temperatures of 40–42°C (104–107°F) in severe cases.	Signs: very hot skin, stopping sweating despite heat, confusion, rapid heartbeat, muscle rigidity. DO NOT wait to see if they cool down. Call 911. Move to cool area immediately. Remove excess clothing. Ice packs to neck, armpits, and groin. Cooling is the treatment — and time matters acutely.
Hyponatremia (water intoxication)	MDMA causes inappropriate antidiuretic hormone (ADH) secretion, causing water retention. People warned about dehydration drink too much water, diluting blood sodium to dangerous levels. The brain swells. This is paradoxically most common in women, who have a higher vulnerability to MDMA-induced hyponatremia.	Signs: severe headache, nausea and vomiting, confusion, seizure — occurring in someone who has been drinking large amounts of water and not urinating adequately. Call 911. Do NOT give more water. Isotonic drinks or medical sodium replacement is required. This is a hospital emergency.

The paradox: too little water (hyperthermia risk) AND too much water (hyponatremia risk) both kill. The correct approach: 500ml (about 16oz) of isotonic fluid per hour during active dancing, or sipping water moderately — not drinking in large volumes. If not dancing and not in intense heat: normal drinking is appropriate.

Adulterant Danger — What Is Sold as MDMA Often Is Not

⚠ THE MAJORITY OF PILLS AND POWDERS SOLD AS MDMA OR ECSTASY IN THE CURRENT SUPPLY CONTAIN SUBSTANCES OTHER THAN OR IN

ADDITION TO MDMA. SOME ADULTERANTS ARE FAR MORE DANGEROUS THAN MDMA ITSELF.

Adulterant	Danger	Detection
PMA / PMMA (para-methoxyamphetamine)	More toxic than MDMA, slower onset (people take more thinking the pill 'is not working'), produces extreme hyperthermia at lower doses. Responsible for many festival deaths attributed to 'ecstasy overdose.'	Marquis reagent: MDMA turns purple/black; PMA turns green/yellow. Testing is essential. If a pill is slow to take effect — do NOT redose before testing.
Fentanyl	Opioid added to many street drugs including MDMA tablets. Even small amounts can cause respiratory depression and death. Particularly dangerous combined with MDMA's hyperthermia because body temperature affects fentanyl's effects.	Fentanyl test strips: dissolve a small amount in water, dip the strip. A positive result means fentanyl is present. Carry naloxone if using any unverified MDMA. Naloxone reverses opioid overdose and does not harm someone who has not taken opioids.
Methamphetamine	Longer-acting, higher cardiovascular risk, much higher addiction liability than MDMA. Many people who believe they are using MDMA are using methamphetamine or an MDMA/meth combination.	Simon's reagent differentiates MDMA (turns blue) from secondary amines like meth (does not turn blue). Marquis reagent: MDMA turns purple/black; meth turns orange/brown.
NBOMe compounds / novel psychoactives	Synthetic psychedelics with narrow therapeutic windows — doses that are recreational can be lethal. Occasionally substituted in MDMA pills.	Reagent testing (Mecke, Marquis) can identify these. Ehrlich reagent specifically detects indoles (including many psychedelics) — if MDMA tests negative on Ehrlich, a psychedelic adulterant may be present.

Reagent testing kits are inexpensive, widely available online, and provide meaningful harm reduction for anyone who has not stopped using. The Marquis, Mecke, Simon's, and Ehrlich reagents together give a comprehensive picture of what is in a pill or powder. Drug checking services at some festivals and through harm reduction organizations provide professional analysis. Fentanyl test strips are available at pharmacies and harm reduction organizations.

Serotonin Syndrome — The Critical Drug Interaction

⚠ MDMA + ANY SEROTONERGIC MEDICATION = POTENTIALLY FATAL SEROTONIN SYNDROME. THIS COMBINATION IS DOCUMENTED TO HAVE KILLED PEOPLE WHO DID NOT KNOW THE RISK.

Serotonin syndrome occurs when serotonergic activity in the nervous system becomes excessive — producing a triad of altered mental status, neuromuscular abnormality, and autonomic instability that can be fatal. MDMA's massive serotonin release combined with any medication that also increases serotonin creates this risk.

- SSRIs (Prozac, Zoloft, Lexapro, Celexa, Paxil): block serotonin reuptake → combined with MDMA's serotonin flooding → serotonin syndrome risk. Also: SSRIs significantly reduce MDMA's subjective effects — people may take more trying to feel the effect, increasing toxicity risk.
- SNRIs (Effexor, Cymbalta, Pristiq): same mechanism as SSRIs with added norepinephrine component.
- MAOIs (phenelzine, tranylcypromine, some foods): block serotonin breakdown → combined with MDMA = acute, severe serotonin syndrome. MAOIs are also found in some supplements (high-dose St. John's Wort) and in harmaline (found in ayahuasca). This combination has killed people.
- Tramadol: mild serotonergic activity — combined with MDMA significantly elevates serotonin syndrome risk.
- Triptans (sumatriptan, rizatriptan — migraine medications): serotonin agonists — risk elevated.

Serotonin syndrome recognition: Agitation, confusion, rapid heart rate, high blood pressure, dilated pupils, muscle twitching, muscle rigidity (especially legs), diarrhea, high body temperature, sweating. Call 911 immediately. Treatment is supportive — cooling, benzodiazepines for agitation, cyproheptadine (a serotonin antagonist) in severe cases.

Section 1 — What MDMA Did to Your Brain

THE CENTURION — The Entactogen — The Drug That Opens the Inside

'Entactogen' means 'producing a touching within' — the pharmacological name for MDMA's classification is more poetic than most drug terminology. Unlike stimulants (which generate external-facing aliveness), depressants (which produce internal quieting), or dissociatives (which remove the self from the room), MDMA produces inward-facing openness: the barriers between the self and other people temporarily dissolve.

This is not metaphor. It is neurochemistry. And understanding which neurochemistry explains why serotonin — not dopamine — is the primary recovery target.

The Four Systems MDMA Activated

System	What MDMA did	What recovery requires
Serotonin (primary — dominant mechanism)	Reversed serotonin transporters (SERT) — forcing massive serotonin release from presynaptic terminals while simultaneously blocking reuptake. The result: 3–4 times more serotonergic activation than dopaminergic. The warm glow, the emotional openness, the sense that everything is all right, the love for the people around you — all primarily serotonergic.	Serotonin system reconstruction is the central biological recovery project. The depletion is specific, significant, and documented on neuroimaging. 5-HTP and L-tryptophan are the primary reconstruction supplements. The social warmth and emotional openness that MDMA produced were serotonergic — rebuilding the serotonin system rebuilds access to these states through natural means. This takes months.
Oxytocin (the bonding molecule)	MDMA triggers oxytocin release from the hypothalamus — the same molecule released during breastfeeding, orgasm, and genuine human bonding. This is why MDMA produces the specific quality of loving closeness rather than simply euphoria. It is the empathy and connection molecule,	The oxytocin system responds to genuine human touch, eye contact, shared experience, authentic vulnerability, and trust-building. Recovery must rebuild the conditions under which the oxytocin system generates its own signal. These conditions are: genuine human connection, physical safety, and the practiced capacity for vulnerability — all of which take time and will to build.

	pharmacologically activated.	
Dopamine (secondary)	MDMA also reverses dopamine transporters (DAT) and norepinephrine transporters (NET), producing secondary stimulant-like effects. The energy, the physical confidence, the dancing endurance are dopaminergic and noradrenergic. Dopamine is amplified by MDMA but at a lower ratio than in cocaine or meth.	D2 receptor downregulation occurs with chronic MDMA use — contributing to the anhedonia and motivational deficit of comedowns and recovery. The dopamine reconstruction protocol from the stimulant guides applies as a secondary track in MDMA recovery, not the primary one.
Serotonin neurotoxicity (chronic high-dose use)	At high doses and with frequent use, MDMA is directly neurotoxic to serotonin-producing axon terminals — the projections from the raphe nuclei to the prefrontal cortex, hippocampus, and striatum. Imaging studies show reduced SERT density (serotonin transporter binding) in heavy users. This represents structural damage beyond receptor downregulation.	Serotonin neurotoxicity from MDMA is partly reversible with sustained abstinence — imaging shows recovery of SERT density over 12–24 months. But the rate of recovery depends on duration and intensity of use, genetic factors (CYP2D6, SLC6A4), and the nutritional and biological support during recovery. The 5-HTP + antioxidant protocol is particularly relevant for reducing the oxidative stress that drives neurotoxicity.

Why MDMA Is Pharmacologically Distinct from Every Other Guide in This Series

THE CENTURION — The Serotonin-First Drug — Not a Stimulant, Not a Depressant, Not a Dissociative

Most guides in this series address either primarily dopaminergic (meth, cocaine) or primarily GABAergic (alcohol, benzos, GHB) or primarily opioidergic (heroin, oxy) substances. MDMA is the only substance in the series whose primary pharmacological mechanism is serotonergic — and this changes the recovery architecture in fundamental ways.

The stimulant guides (meth,	MDMA	Why the difference matters for recovery

cocaine)		
Primary mechanism: dopamine flooding → D2 receptor downregulation → anhedonia	Primary mechanism: serotonin flooding → SERT downregulation and axonal damage → emotional flatness, social disconnection	Tyrosine (dopamine precursor) is the primary stimulant recovery supplement. In MDMA recovery, 5-HTP (serotonin precursor) is primary. Tyrosine is a secondary supplement. Getting this wrong — treating MDMA recovery like stimulant recovery — produces incomplete biological reconstruction.
What was lost: aliveness, motivation, intensity, performance	What was lost: emotional warmth, social ease, the felt sense of connection and love, empathy	The recovery target is different. Stimulant recovery builds intensity and motivation. MDMA recovery builds connection and emotional warmth. The natural addiction architecture is completely different: genuine human connection rather than competition and stimulation.
Psychiatric risk: stimulant psychosis, paranoia	Psychiatric risk: MDMA-induced depression, depersonalization, anxiety, emotional blunting	The psychiatric picture in MDMA recovery is serotonergic depression and social anxiety — not the paranoid hypervigilance of stimulant recovery. SSRIs and SNRIs treat serotonergic depression — but their interaction with residual MDMA requires monitoring. 5-MTHF is often transformative for the MTHFR-variant subset.

The MDMA Cycle — Use, Depletion, Comedown, Redosing

THE CENTURION — The Compendium Names the Pattern: The Social API Collapse

The MDMA use pattern follows a predictable pharmacological cycle that the compendium documents in its Phase 1–5 social API collapse framework. Understanding the cycle is understanding why what started as a social enhancement eventually became an isolation mechanism.

Phase	What was happening	The underlying biology
Phase 1 — Social use	MDMA used in community contexts: festivals, raves, parties. The drug enhanced genuine social experiences — music felt	Low frequency use allows serotonin system recovery between uses. Social context means the oxytocin and endorphin co-release of

	transcendent, connections felt profound, the dancing crowd felt like family. This was real. The neurochemistry was real. The relationships formed in this context were real.	genuine human contact accompanies the MDMA serotonin flood. The experience is the synergy of drug and genuine community.
Phase 2 — The honeymoon tightening	Use becomes more frequent. The community context may remain but the drug is being used more intentionally for its effect rather than as part of shared ritual. The compendium: 'the brain noticed that the DRUG provides the dopamine; the PEOPLE are just the delivery mechanism.'	Tolerance beginning to develop. Higher doses needed for the same effect. SERT downregulation starting. The depletion period (the Tuesday blues) is becoming more pronounced — motivating more frequent use to avoid the crash.
Phase 3 — Social decoupling	The ritual with friends becomes the private use. The compendium: 'friends = latency; socializing = overhead; solo use = direct server access.' The substance is now used alone or in smaller, increasingly specific contexts rather than in community.	SERT downregulation is significant. The serotonin available for normal social interaction is depleted. Paradoxically: the drug that was opening the person to connection is now required to feel connected at all — the baseline social capacity has been undermined.
Phase 4 — Isolation and dependence	Solo use is the primary pattern. The compendium: 'now you're alone wondering why you don't want to see anyone, why Netflix feels pointless, why your only comfortable space is behind a screen.' The social drug has produced complete social withdrawal.	Serotonin system significantly depleted and possibly neurotoxically damaged. Oxytocin system reduced in sensitivity. The person cannot generate adequate serotonin for comfortable social interaction without the drug — which was the thing destroying their ability to generate serotonin.
Phase 5 — Recovery entry	This is where you are. The substance that opened the person to connection has isolated them from it. The work of recovery is rebuilding the serotonin system and the social capacity simultaneously — one feeding the other.	Serotonin system recovery begins with abstinence. It is supported by 5-HTP, exercise, sunlight, and genuine social contact — which directly activates the same oxytocin and serotonin pathways that MDMA was activating pharmacologically.

The Tuesday Blues — The Pharmacology of the Comedown

THE CENTURION — Not Weakness. Serotonin Depletion.

The MDMA comedown — arriving typically 1–3 days after use, hence 'Tuesday blues' (Monday use at a weekend festival) — is one of the most precisely documented substance after-effects in pharmacology. It is serotonin depletion: the massive release MDMA forced from presynaptic terminals takes 48–72 hours to synthesize and restore from dietary tryptophan.

During this period, the brain has less available serotonin than before the use. The emotional consequences are depression, anxiety, emotional sensitivity, social withdrawal, fatigue, and the overwhelming sense that ordinary life is gray compared to the state the MDMA produced. For the person with an already-low serotonin baseline — SLC6A4 short/short allele, MTHFR-impaired synthesis, or chronic stress-depleted system — the Tuesday blues may be severe enough to mimic clinical depression.

Understanding that the Tuesday blues is a scheduled pharmacological consequence — not evidence that sober life is genuinely inadequate — is the first cognitive reframe that changes the relationship to the cycle. The 5-HTP protocol (Section 4.2) can significantly reduce comedown severity. Using 5-HTP after MDMA to accelerate serotonin recovery is documented in harm reduction literature and referenced in the compendium's supplement table.

Section 2 — Your Genomic Blueprint

MDMA has a genomic profile as specific as any substance in this series — but oriented toward the serotonin system rather than the dopamine system. Understanding these variants explains both why MDMA felt necessary and which recovery approaches will be most effective.

The DNA Testing Panel for MDMA Recovery

Gene	What it controls	Key variant	MDMA recovery relevance
CYP2D6	Primary metabolizer of MDMA — converts MDMA to its active metabolites and ultimately to inactive compounds for clearance	*4/*4 poor metabolizer (7–10% Caucasian), *1/*4 intermediate, various ultra-rapid variants	THE most critical safety gene for MDMA. The compendium explicitly names CYP2D6 as critical for empathogens like MDMA. Poor metabolizers (*4/*4) cannot clear MDMA adequately — it accumulates to toxic levels at doses that would be manageable for normal metabolizers. This dramatically increases serotonin syndrome risk, hyperthermia risk, and neurotoxicity risk. If you have ever experienced MDMA effects dramatically more intense or longer than others at the same dose — you may be a CYP2D6 poor or intermediate metabolizer. This information is critical for future harm reduction and for understanding why the same dose damaged you more.
SLC6A4 / 5-HTTLPR	Serotonin transporter expression — the primary target of MDMA's mechanism	Short/short, long/long, S/L heterozygous alleles	The serotonin transporter gene directly determines who is most vulnerable to MDMA's serotonergic effects and most susceptible to serotonin depletion with use. Short/short (SS) allele carriers have reduced SERT expression — lower baseline serotonin transporter density — meaning MDMA's SERT reversal produces a more profound serotonergic surge AND a more severe depletion state. SS carriers: higher MDMA response, more severe Tuesday blues, more severe long-term depletion. The compendium: MDMA addresses 'serotonin, social bonding deficit' — SS carriers are

			most likely to have this deficit.
MTHFR	Methylation — serotonin synthesis foundation	C677T (rs1801133), A1298C (rs1801131)	Serotonin synthesis requires folate — specifically, the folate-dependent methylation cycle produces the BH4 (tetrahydrobiopterin) cofactor essential for tryptophan hydroxylase (the enzyme that converts tryptophan to 5-HTP and then to serotonin). MTHFR variants that impair this process create a baseline serotonin synthesis deficit — the foundation deficit that MDMA was temporarily filling. 5-MTHF supplementation addresses this mechanism directly. Many people with chronic MDMA use disorder who also have MTHFR variants find 5-MTHF to be transformative in recovery — often the most impactful single supplement.
TPH2	Tryptophan hydroxylase 2 — the rate-limiting enzyme in serotonin synthesis in the brain	rs4570625 (T/T vs. G/G)	TPH2 variants affect the speed and efficiency of serotonin synthesis from tryptophan — directly affecting both the serotonin baseline and the rate of recovery after MDMA depletion. Low-activity TPH2 variants produce slower serotonin recovery from depletion — meaning longer comedowns and slower long-term recovery. 5-HTP bypasses TPH2 entirely (it enters the pathway after the rate-limiting step), which is why it is preferred over L-tryptophan for rapid serotonin recovery.
COMT	Dopamine and stress chemical clearance speed	Val158Met (rs4680)	COMT affects MDMA recovery through the dopamine secondary mechanism. Val/Val (Warrior) carriers who also used MDMA — particularly in chemsex contexts or alongside stimulants — were driven to MDMA partly by its dopamine component alongside its serotonergic connection effect. Recovery architecture for Val/Val carriers needs to address both the serotonin reconstruction AND the Warrior's intensity requirement. Met/Met (Sage) carriers are more likely to have been using MDMA primarily for its serotonergic anxiolytic and social-ease effects.
HTR2A / HTR2B	Serotonin 5-HT2A and 5-	Multiple variants	5-HT2A receptor variants affect the intensity of MDMA's hallucinogenic

	HT2B receptor genes — primary postsynaptic serotonin receptors activated by MDMA	affecting receptor sensitivity	and empathogenic component — people with more sensitive 5-HT2A receptors experience more intense MDMA effects at the same dose. 5-HT2B downregulation with chronic use contributes to the emotional blunting of heavy MDMA use. Receptor sensitivity recovery is supported by abstinence and omega-3 DHA supplementation.
BDNF	Brain-derived neurotrophic factor — serotonin axonal repair mechanism	Val66Met (rs6265)	MDMA's serotonin neurotoxicity damages serotonergic axon terminals — the structures that release serotonin into synapses. Recovery of these terminals requires BDNF-driven neuroplasticity. Met carriers have reduced BDNF secretion, potentially slowing the recovery of neurotoxically damaged serotonin axons. Exercise is the primary BDNF driver and becomes critical for axonal repair in heavy MDMA users.

Phenotyping Without Test Results

- Did MDMA affect you much more intensely than others at the same dose, or did the effects last dramatically longer? → Likely CYP2D6 poor or intermediate metabolizer. Your total MDMA exposure and neurotoxicity risk is higher than equivalent dose users with normal CYP2D6.
- Were your Tuesday blues consistently more severe than those of people you used with at similar doses? → Likely SLC6A4 SS allele (reduced baseline SERT) compounding depletion effects, or CYP2D6 slow metabolism producing higher exposure, or MTHFR-variant impaired serotonin synthesis capacity.
- Did MDMA seem to specifically address a social anxiety or social disconnection that felt chronic before you started using? → SLC6A4 SS allele plus MTHFR variant is the most common combination underlying this experience. The serotonin deficit was real. The MDMA was solving a genuine biological problem.
- Have you been diagnosed with or suspected of having depression that did not adequately respond to SSRIs? → MTHFR involvement is likely. 5-MTHF supplementation should be assessed before any new SSRI trial.
- Was MDMA used primarily in community and festival contexts for its social and empathogenic properties, not for stimulant effects? → The serotonin and oxytocin systems are the primary recovery targets. Dopamine reconstruction is secondary.

Section 3 — The Detox Protocol

THE CENTURION — MDMA Withdrawal Is Not Medically Dangerous — But the Serotonin Floor Is Real

MDMA does not produce the seizure risk of alcohol or benzodiazepines. It does not produce the cardiovascular emergency of acute cocaine use. Physical withdrawal from MDMA — in the sense of a medical emergency requiring supervised detox — does not exist in the way it does for most substances in this series.

What MDMA does produce — particularly with frequent or heavy use — is a protracted serotonin deficit state that can be severe enough to meet clinical criteria for major depressive disorder. For heavy users with underlying serotonin synthesis vulnerabilities (MTHFR, SLC6A4, TPH2 variants), stopping MDMA can unmask a depression that requires medical management in its own right.

The acute medical cautions in Section 0 are for during MDMA use, not for withdrawal. Once MDMA is stopped, the primary risks are psychiatric rather than medical.

The Withdrawal Timeline

Timeline	Biology	Symptoms	What to do
Days 1–3 (the extended comedown)	Serotonin reserves depleted by most recent use; synthesis rate of tryptophan → 5-HTP → serotonin inadequate to meet the demand created by the depleted baseline	Depression, fatigue, anxiety, social withdrawal, emotional sensitivity, insomnia or hypersomnia, poor concentration — the Tuesday blues in extended form	This is the pharmacological comedown, not 'withdrawal.' 5-HTP (100mg, 2–3 days after last use) accelerates serotonin recovery. Rest, hydration, nutrition (especially tryptophan-rich foods: turkey, eggs, dairy, nuts). Do not use MDMA to end the comedown — this is the primary trap that drives the cycle.
Week 1 (the serotonin floor)	Baseline serotonin synthesis attempting to recover to pre-MDMA levels; but if use was frequent and/or neurotoxic damage occurred, the 'pre-MDMA level' may itself have been depleted	Persistent low mood, social disconnection, emotional flatness, possible depersonalization or derealization (feeling unreal), impaired memory and concentration, loss of interest in activities that previously	Start full supplement protocol. Begin exercise immediately — BDNF from exercise supports serotonin axonal repair. Sunlight and circadian reset (morning light exposure). Social contact — even brief, low-demand contact — activates the oxytocin system and supports serotonin.

		generated pleasure	
Weeks 2–8	Serotonin system recovering; 5-HT2A and 5-HT2B receptor sensitivity gradually increasing; SERT density beginning to recover; BDNF supporting axonal repair where damage occurred	Gradual improvement in mood with significant variability; emotional range slowly widening; social connection becoming more accessible; cognitive function improving; sleep slowly normalizing	Supplement protocol fully underway. Exercise daily. Sunlight daily. Social engagement building — start small and authentic. Track mood variably — the recovery is not linear and improvement can be obscured by the variability.
Months 1–6	Significant serotonin system recovery; oxytocin system resensitized; dopamine secondary recovery underway; BDNF-driven neuroplasticity rebuilding serotonergic axons	For most users with recreational patterns: substantially recovered mood, social capacity, and emotional warmth. For heavy, long-term users with neurotoxic damage: ongoing but measurable recovery; some residual emotional blunting that resolves over 12–24 months.	This is when recovery starts to feel genuinely different from early recovery. Emotional range returns. Genuine connection begins to feel possible without chemicals. The natural connection architecture (Section 6) becomes increasingly effective.
Months 6–24 (neurotoxic recovery)	For heavy users with documented SERT density reduction: gradual recovery of serotonergic axon terminal density; most studies show substantial but not always complete recovery at 12–24 months sustained abstinence	Progressive resolution of residual emotional blunting; cognitive function normalized; social emotional range fully restored in most	Imaging studies show SERT density recovery at 12–24 months. This is not speculative — it is documented. The brain repairs itself. The timescale is longer than the subjective experience of improvement, which occurs earlier.

The Depression Emergency

⚠ FOR PEOPLE WITH SIGNIFICANT MDMA USE HISTORY AND CO-OCCURRING SEROTONERGIC DEPRESSION: STOPPING MDMA MAY UNMASK SEVERE DEPRESSION. HAVE A PSYCHIATRIC PLAN IN PLACE BEFORE STOPPING IF THIS APPLIES TO YOU.

The person who used MDMA regularly to feel emotionally normal, socially connected, or adequate may find that stopping MDMA reveals a depressive or anxiety disorder that was being managed by the drug. This is not evidence that MDMA is needed permanently. It is evidence that the underlying condition requires its own treatment.

The 5-MTHF assessment should occur immediately: if MTHFR variants are likely (prior SSRIs didn't work well; folate supplementation seems to help), start 5-MTHF before considering prescription antidepressants. Many people who did not respond to SSRIs respond to 5-MTHF — because the barrier to their serotonin recovery was upstream (impaired methylation) rather than in the reuptake mechanism that SSRIs address.

Section 4 — Biological Reconstruction

MDMA recovery's biological reconstruction has a single primary target — the serotonin system — with dopamine as a secondary target and the methylation foundation underlying both. The oxytocin system is addressed primarily through behavioral means (genuine human connection) rather than supplements, though some supporting compounds exist.

4.1 — The Methylation Foundation

Serotonin synthesis is folate-dependent through the BH4 pathway. MTHFR variants impair the conversion of dietary folate to the active methylfolate needed for this pathway — creating a serotonin synthesis bottleneck that MDMA was temporarily bypassing by forcing release of whatever serotonin was available. 5-MTHF addresses the bottleneck directly.

Supplement	Dose	Timing	MDMA-specific notes
5-MTHF (L-methylfolate)	400–1000mcg daily	Morning with food	The compendium lists 5-MTHF first in the methylation support table specifically because serotonin synthesis is folate-dependent. For MDMA recovery with MTHFR variants, 5-MTHF often produces the most dramatic early improvement — because the serotonin synthesis rate was impaired at the methylation level, not just the precursor level. If you have tried 5-HTP without adequate improvement, add 5-MTHF and assess response.
Methylcobalamin (B12)	1000–2000mcg sublingual	Morning	Works synergistically with 5-MTHF in the methylation cycle. MDMA use associated with the festival lifestyle (poor sleep, poor nutrition, alcohol co-use) depletes B12. Sublingual methylcobalamin. Also supports neurological integrity relevant to the serotonergic neurotoxicity recovery.
P5P (active B6)	25–50mg	Morning	Cofactor for aromatic amino acid decarboxylase — the enzyme converting 5-HTP to serotonin. Without adequate P5P, the 5-HTP

			supplementation below cannot efficiently convert to serotonin. This is the enabling cofactor for the serotonin reconstruction stack. Use P5P form — not standard pyridoxine.
Magnesium glycinate	400–600mg elemental	Morning and evening	Cofactor for multiple enzymes in the methylation cycle and serotonin synthesis pathway. Anxiety reduction support. Sleep improvement. MDMA use and the associated lifestyle factors reliably deplete magnesium. The anxiety and sleep disruption of MDMA recovery respond specifically to magnesium.

4.2 — Serotonin Reconstruction — The Primary Target

THE CENTURION — The Compendium's Table Is Exact: Serotonin Is Depleted by MDMA; 5-HTP Is the Reconstruction Supplement

⚠ DO NOT COMBINE 5-HTP WITH ANY SEROTONERGIC MEDICATION WITHOUT PHYSICIAN GUIDANCE. SSRIs, SNRIs, MAOIs, TRAMADOL, AND TRIPTANS ALL CARRY SEROTONIN SYNDROME RISK IN COMBINATION WITH 5-HTP. THIS IS A REAL DANGER, NOT A THEORETICAL ONE.

Supplement	Dose	Timing	MDMA-specific notes
5-HTP (5-hydroxytryptophan)	50–200mg daily (titrate up slowly)	Evening primarily; or split AM/PM	The compendium names 5-HTP as the primary serotonin reconstruction supplement for MDMA depletion. 5-HTP bypasses the rate-limiting TPH2 step in serotonin synthesis, providing direct serotonin precursor that the depleted system can immediately use. Start at 50mg and increase by 50mg every 1–2 weeks assessing response. Too high a dose too quickly can cause gastrointestinal distress or, in sensitive individuals, overshooting the serotonin target and causing anxiety. Take with P5P (the cofactor for conversion). Do not combine with SSRIs, MAOIs, or other serotonergic medications.

L-Tryptophan (alternative to 5-HTP)	500–2000mg	Evening, away from protein meals	The compendium notes: use one or the other — do not combine 5-HTP and L-tryptophan. L-tryptophan goes through the full synthesis pathway (tryptophan → 5-HTP → serotonin via TPH2), making it slower-acting but more physiologically regulated. Some people find tryptophan gentler and less likely to overshoot. For those with low-activity TPH2 variants, 5-HTP is preferred (bypasses the slow enzyme).
EGCG (green tea extract)	400–800mg standardized	Morning	A specific addition for MDMA recovery: EGCG (epigallocatechin gallate) has been studied specifically for MDMA-related neuroprotection in animal models, reducing the neurotoxic damage to serotonin axon terminals. The antioxidant mechanism protects against the oxidative stress that drives MDMA's serotonergic neurotoxicity. Start early in recovery and continue for the first 6 months.
Alpha-lipoic acid	300–600mg daily	Morning, away from food	Potent antioxidant that crosses the blood-brain barrier — particularly relevant for MDMA's oxidative damage to serotonin axon terminals. Regenerates vitamin C, vitamin E, and glutathione simultaneously. The combination of alpha-lipoic acid + EGCG + NAC provides comprehensive antioxidant coverage for MDMA neurotoxicity recovery.
NAC (N-Acetyl Cysteine)	600–1800mg daily in divided doses	Morning and evening with food	Glutathione precursor — glutathione is the brain's primary antioxidant for protecting against MDMA's oxidative neurotoxicity. NAC restores glutathione in the brain and simultaneously restores glutamate homeostasis (relevant for the dopamine secondary recovery). One of the most multi-functional supplements for MDMA recovery.
Omega-3 DHA + EPA	2–4g daily (EPA-	With food, morning	DHA supports serotonin receptor membrane environment and 5-

	dominant for mood)		HT2A receptor function. EPA has anti-inflammatory and mood-supporting effects relevant to the serotonergic depression of MDMA recovery. The 5-HT2A receptors damaged by chronic MDMA use are embedded in neuronal membranes — DHA's membrane-supporting role is directly relevant.
Inositol	4–12g daily (titrate)	Divided with meals	Modulates 5-HT2A receptor sensitivity — relevant as these receptors recover from MDMA-induced downregulation. Also has evidence for anxiety and OCD — both common in MDMA recovery. The social anxiety that MDMA was managing (and that is now unmedicated) responds specifically to inositol's serotonin receptor modulating effects.

4.3 — The Neuroprotection Stack

THE CENTURION — Protecting the Repair While It Is Happening

For people with heavier MDMA use histories — multiple times per month over years, or high-dose single use — the serotonin axon terminals may have sustained oxidative damage. The neuroprotection stack reduces ongoing oxidative stress and supports the BDNF-driven repair of these terminals.

- Vitamin C (1000–2000mg daily, divided): MDMA depletes vitamin C specifically through oxidative stress mechanisms. Vitamin C is a direct antioxidant in the serotonergic synapse. Deficiency worsens MDMA-related neurotoxicity. Replenishment is particularly important in the first month of recovery.
- Vitamin E (400 IU, mixed tocopherols, daily with food): fat-soluble antioxidant that protects neuronal membranes from the lipid peroxidation that MDMA's oxidative stress produces. Mixed tocopherols (not alpha-tocopherol alone) for full protective coverage.
- Melatonin (low dose, 0.5–1mg before bed): melatonin has specific neuroprotective properties against MDMA-induced serotonergic neurotoxicity in research models — beyond its sleep-supporting role. This is an additional reason to include low-dose melatonin in MDMA recovery beyond sleep architecture normalization.
- CoQ10 (100–200mg daily): mitochondrial antioxidant; protects against the mitochondrial oxidative stress that MDMA's hyperthermia and metabolic

demands produce; relevant for any MDMA use that involved significant physical exertion (dancing for hours).

4.4 — Dopamine Secondary Recovery

MDMA's secondary dopamine effects require secondary dopamine reconstruction — but at a lower priority than the serotonin primary track. The dopamine reconstruction supplements from the meth and cocaine guides apply here, at more moderate doses:

- L-Tyrosine (500–1000mg, morning, away from protein): dopamine precursor. Lower priority and lower dose than in pure stimulant recovery. Add after the serotonin stack is established.
- Uridine (250mg) + DHA (1g) + CDP-choline (250mg) — the Brain Stack: D1/D2 receptor density restoration. The secondary dopamine receptor downregulation from MDMA's dopamine component responds to this combination.
- Rhodiola rosea (300mg, morning): dopamine and serotonin modulation; adaptogen; anti-fatigue. Useful in weeks 2–8 when low motivation and energy are compounding the serotonin flatness.

4.5 — Sleep Reconstruction

MDMA significantly disrupts sleep architecture — suppressing REM sleep during use, often followed by hypersomnia during the comedown. For frequent users, the cumulative REM debt affects serotonin production (serotonin is synthesized during specific sleep stages) and creates a cycle of poor sleep → impaired serotonin synthesis → worse mood → desire to use.

- Melatonin: 0.5–1mg sublingual, 30 min before bed. Low dose — melatonin is synthesized from serotonin; supporting the serotonin synthesis (above) supports melatonin production indirectly.
- Magnesium glycinate: 400mg evening. Anxiety reduction; GABA support for sleep onset.
- Glycine: 3–5g, 30 min before bed. Core body temperature reduction; NMDA co-agonism.
- 5-HTP (if not taken in morning): 50mg evening. Serotonin-melatonin conversion supports both mood and sleep.
- Tart cherry extract: 480mg evening. Natural melatonin precursor source; anti-inflammatory.

The vivid, emotionally intense dreams of early MDMA recovery are REM rebound — the brain claiming the serotonin-dependent sleep stages that MDMA was

suppressing. They can be overwhelming and emotionally intense. They are healing. They are the emotional memory processing that MDMA was preventing.

4.6 — Peptide Support Layer

Peptide	Mechanism in MDMA recovery	Timing	Notes
BPC-157	Dopamine AND serotonin system modulation; gut-brain axis repair; neuroprotection from oxidative injury; BDNF expression support — the multi-system coverage directly relevant to MDMA's multi-system damage	Day 1 post-cessation — daily	BPC-157's serotonin system modulating properties make it particularly relevant for MDMA recovery — the serotonin and dopamine concurrent effects address both primary and secondary MDMA damage tracks. The gut-brain axis repair is relevant given MDMA's significant effects on gut serotonin (90% of serotonin is in the gut — GI symptoms during comedowns reflect gut serotonin depletion). Oral or subcutaneous.
Selank	GABA and serotonin modulation; anxiolytic without dependence — the social anxiety of MDMA recovery specifically responds to Selank's serotonergic component	Weeks 1–8 as needed for social anxiety	For the person whose MDMA use was managing chronic social anxiety, Selank provides non-addictive serotonergic anxiolytic support during the recovery period when the anxiety is unmedicated and the serotonin system is still rebuilding. Intranasal.
Semax	BDNF and NGF upregulation — critical for serotonergic axonal repair in neurotoxic MDMA use; cognitive enhancement; serotonin and dopamine modulation	Week 2 onwards	For heavy MDMA users where serotonin axonal damage has occurred, Semax's BDNF-stimulating effects directly support the repair mechanism. The cognitive fog and memory impairment of significant MDMA history responds to Semax's prefrontal support. Intranasal.
Epitalon	Sleep architecture normalization; pineal pathway; circadian reset — melatonin synthesis	Weeks 1–4 post-cessation	Melatonin is synthesized from serotonin — the same system MDMA depleted. Epitalon supports the pineal

	from serotonin is directly relevant to MDMA recovery		pathway and circadian biology that depends on adequate serotonin. Particularly useful in the first month when sleep disruption is compounding the serotonin recovery.

Section 5 — The MDMA Craving — Understanding and Interrupting

THE CENTURION — The Craving Is for the Connection. Everything Else About MDMA Is Background.

MDMA cravings have a specific quality that separates them from every other craving in this series. They are not primarily for the euphoria (though that is present). They are not primarily for the stimulant energy (though that contributes). They are cravings for a specific quality of connection — the feeling that barriers between self and other people are dissolved, that genuine contact is possible without self-consciousness, that the people around you are exactly where they should be and so are you.

This means: the most powerful MDMA triggers are not the sight of powder or a pill. They are social situations where connection feels inaccessible — awkward social events, parties where the person feels invisible, moments of profound loneliness in crowds, situations where they feel they cannot adequately present their authentic self.

The interrupt must address the connection deficit, not simply block the substance craving.

MDMA Craving Trigger Map

Trigger	The underlying state	The interrupt
Social events — especially events associated with MDMA use	The environmental cue activates the anticipatory serotonin and oxytocin circuit. Festival music, electronic music, nightclub environments, rave aesthetics — any of these fire the conditioned craving with high intensity.	Pre-plan high-risk social events: arrive with a sober support person; identify an exit plan; take your supplement stack before attending; limit time in the highest-intensity cue environments early in recovery. Protocol 05 (environmental redesign) for the most high-risk contexts.
Feeling socially disconnected or invisible	The core state MDMA was addressing: 'I am in a room full of people and I cannot make genuine contact with any of them.' The craving fires as the solution the nervous system learned for this specific problem.	This is the craving that requires the most sophisticated response because it is pointing at a real unmet need. Contact with one person directly — not the party, the one person. A genuine exchange, however brief. The oxytocin system responds to direct eye contact, authentic conversation, and physical

		presence. These are the natural tools.
Festival music / specific artists / specific tracks	Auditory conditioning: specific music was paired with MDMA's peak experience hundreds of times. The music fires the anticipatory dopamine and serotonin signals before conscious awareness of the craving. This is exceptionally powerful and often arrives as emotion before it arrives as thought.	Build a recovery playlist that is genuine and meaningful — music that is not MDMA-associated, that activates serotonin and oxytocin through its own qualities (Protocol 12, Sonic Architecture in the main Compendium). The music on that playlist becomes the new auditory home. The MDMA music can be integrated later — but not in early recovery.
Contact from former using community	A message from a festival friend, an invitation to an event, a photo from a past experience — activates the full conditioned response: social belonging, chemical anticipation, the memory of connection.	This is the social cost of early MDMA recovery that is most underaddressed. The festival community was the social world. Protecting early recovery sometimes requires distance from that world while the serotonin system and the capacity for sober connection rebuild. This is temporary. It is not permanent exile.
The Tuesday blues / depression wave	The come-down state, even weeks after last use, can fire MDMA craving as the most reliable available solution for the depression it is producing. 'I just want to feel normal again' — and MDMA is the fastest available return to feeling normal.	Name the Tuesday blues as a biological event, not a life verdict. Execute the full serotonin support stack (additional 5-HTP, sunlight, social contact, exercise). Contact your support structure. The compendium: the comedown is the pharmacological consequence, not the truth about your life.

THE MDMA CRAVING INTERRUPT — 5-MINUTE PROTOCOL

1. NAME THE SPECIFIC CRAVING. Not 'I want to do MDMA.' What is the state underneath it? Social disconnection? Tuesday blues depression? Music-triggered anticipation? Festival nostalgia? Identifying the specific driver changes the response.
2. EXECUTE 4-7-8 BREATHING — SIX CYCLES. The serotonin-oxytocin craving fires in the limbic system before the prefrontal cortex can evaluate it. Extended exhale breathing activates the vagus nerve and the parasympathetic system — creating the gap between impulse and response in which the prefrontal cortex can participate.
3. TAKE 5-HTP AND MAGNESIUM. The serotonin precursor and the cofactor — immediately. The craving has a biological substrate (serotonin deficit driving

the desire for serotonin flooding) and the supplement directly addresses that substrate. This is not a placebo — it is precursor provision.

4. MAKE GENUINE CONTACT WITH ONE PERSON. Not a text to the group chat. Not social media. One person — call them or meet them — and have one genuine exchange. Look at them. Listen to them. This activates the oxytocin system through the mechanism it was designed for: direct human contact. The MDMA craving for connection is best interrupted by real connection.
5. EXECUTE YOUR NATURAL SEROTONIN ACTIVATION. From Section 6: genuine social engagement in a context that feels real, laughter, movement with others, creative expression with an audience, service that is genuinely useful to someone. The natural serotonin-oxytocin architecture provides the state the craving is seeking.
6. ASSESS THE SOCIAL CONTEXT. If the craving was triggered by a social environment — is this environment appropriate for early recovery? Protocol 05 provides the environmental redesign framework. Some environments are too high-risk in the first 6 months regardless of the person's intention.
7. DOCUMENT. Trigger, underlying state, intensity (0–10), action, outcome. Three weeks of this documentation reveals the specific conditions under which the connection deficit activates the MDMA craving — and therefore reveals exactly what the natural connection architecture must address.

Section 6 — Porting the Addiction: Building the Natural Connection Architecture

THE CENTURION — Centurian Paper C-28 Applied to MDMA Recovery — The Connection the Serotonin System Was Built to Generate

The compendium's mapping is exact: MDMA addresses 'connection, love, openness' through 'serotonin, social bonding deficit.' The replacement is 'authentic connection practices, community.'

This is the most specific and most honest description of any substance's recovery architecture in this entire series. MDMA recovery is not about finding an alternative to connection. It is about building the conditions under which genuine serotonergic and oxytocinergic connection occurs naturally.

Your serotonin system knows how to generate the connection state. It was designed for it. MDMA was the shortcut to a destination the nervous system already knows how to reach. Recovery is building the road rather than borrowing the car.

The Natural Serotonin and Oxytocin Architecture

Practice	Primary molecule	How to implement	For MDMA recovery specifically
Genuine deep conversation — one person, authentic exchange	Oxytocin + serotonin — both released in genuine relational connection	Schedule regular one-to-one time with trusted people. Lower the social mask deliberately. Practice authentic disclosure — small at first, growing with trust.	This is the most direct natural replacement for MDMA's oxytocin release. MDMA's social magic was the removal of the barrier between self and other — the feeling that genuine contact was happening without the protective performance. Building the capacity for this in sober conversation, with one person at a time, in lower-stakes contexts first, is the central social recovery practice.
Physical safe touch	Oxytocin — primarily released through safe physical contact	Hugs, physical presence, safe non-sexual touch with trusted people. Massage. Pets. Physical community activities (yoga, martial arts, dance).	MDMA's tactile dimension — the enhanced sensitivity to touch and the oxytocin-mediated warmth of physical closeness — is partly addressable through safe physical contact in sober contexts. The oxytocin release from a genuine hug is real. It is not the same intensity as MDMA's pharmacological

			oxytocin flood — but it builds the system rather than depleting it.
Communal movement — dance, exercise with others	Serotonin + endorphin + oxytocin — the triad released in synchronized movement with others	Dance classes, group exercise, martial arts, team sports, synchronized activities. With other people — not alone.	Synchronized movement with others (the shared beat of dance, the synchrony of crew rowing, the rhythm of a yoga class) is one of the most reliable natural generators of the social-bonding neurochemistry that MDMA was providing pharmacologically. The festival experience was partly the music and partly the synchronized movement with hundreds of people. Sober movement communities provide the movement and synchrony without the chemical.
Music — live performance and genuine listening	Serotonin + dopamine — music 'chills' involve serotonin and dopamine simultaneously	Attend live music. Listen deeply, actively, to music that genuinely moves you. Make music if possible. The connection to music is real and does not require MDMA.	The relationship between MDMA and electronic music is one of the most specific substance-cultural pairings in modern history. The music was genuinely enhanced by MDMA — but the relationship with music can be rebuilt on its own terms. Live music, active listening, creative music-making: these activate the serotonin-dopamine music response without the pharmaceutical enhancement. The music is still profound sober. It is different, not less.
Laughter and shared joy	Serotonin + endorphin + oxytocin — the triad of genuine social laughter	Genuine comedy. Absurdity. Play. Situations where laughter is the natural response. Scheduled with people who produce it.	The MDMA state included a quality of delight and joy in ordinary things — the feeling that everything was exactly as it should be and was worth celebrating. Genuine laughter activates the same neurochemical triad. It cannot be scheduled directly — but the conditions that produce it can be created: safe people, permission for absurdity, removal of self-seriousness. Recovery needs to include enough laughter to signal that the serotonin system is working.
Service and genuine contribution	Oxytocin + serotonin + dopamine — meaning	Volunteer work, peer recovery support, caregiving, any activity where one's	The MDMA experience included a specific quality of feeling that the presence of others mattered and that your presence mattered to

	activates all three	presence genuinely improves someone else's experience.	others. Genuine service activates this same quality — the felt sense that one's existence is meaningful to someone else — through the oxytocin and serotonin systems that MDMA was pharmacologically activating. For many people in MDMA recovery, service to others becomes the most reliable source of the open-hearted presence they were seeking in the drug.
Sunlight and circadian entrainment	Serotonin — sunlight directly drives serotonin synthesis through the retina-raphe pathway	20+ minutes of direct morning sunlight daily. Not through windows. Outside, within 30–60 minutes of waking.	This is not optional light therapy. The raphe nuclei — the serotonin-producing brain regions that MDMA depleted — receive direct input from the retinohypothalamic tract. Morning light activates this pathway and directly drives serotonin synthesis. For people with serotonin system deficits, morning sunlight is a genuine biological intervention, not a wellness suggestion. Schedule it before anything else in the morning.

Rebuilding the Festival Community — What Was Real, What Can Be Kept

THE CENTURION — The Scene Gave You Something Real. The Question Is What Parts of It Were the Drug and What Parts Were You.

The festival and rave community that MDMA was embedded in was not simply a drug delivery mechanism. It was a genuine community — with shared values (PLUR: Peace, Love, Unity, Respect), shared aesthetic sensibilities, shared music that genuinely moved people, and genuine relationships that formed in the context of shared experience.

Some of that community was chemical. The depth of connection that MDMA produced — the sense that strangers were family, that barriers between people were imaginary — was partly pharmacological and would not have felt the same sober. But some of it was real: real music, real shared experience, real relationships that grew from the scenes they started in.

Recovery does not require leaving the culture entirely — but it requires renegotiating the relationship to the culture. Some environments are too high-risk in early recovery regardless of the person's intention — the cue-reactivity is too strong, the social pressure is too present, and the supplement stack cannot fully

counteract the conditioned neural response. These are managed with Protocol 05 (environmental redesign) and time.

What can often be kept: the music, in sober contexts. The friendships, renegotiated on sober terms. The values — PLUR is not a drug value, it is a human value. The aesthetic. The sense of belonging to a community that sees the world differently.

What requires fundamental renegotiation: any social space where MDMA use is active and expected. Any friend group where the shared social currency is the substance. Any relationship that exists only in the context of use.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	MDMA-specific notes
Biological Foundation (Days 1–14)	Protocol 07, Protocol 09, Protocol 17, Protocol 18	Serotonin reconstruction stack; methylation foundation; sleep; nutrition; neuroprotection antioxidants	5-MTHF, magnesium, and NAC on Day 1. 5-HTP begins 2–3 days after last MDMA use (to avoid any residual interaction risk). P5P before adding 5-HTP. The serotonin reconstruction timeline begins immediately with the methylation foundation, then adds the precursor (5-HTP) once the synthesis cofactors are in place.
Genomic Mapping (Weeks 1–4)	Section 15 (Compendium), CYP2D6, SLC6A4, MTHFR, TPH2	Metabolizer status; serotonin transporter variants; methylation; synthesis rate	CYP2D6 is the highest priority genetic test — it explains why the MDMA experience was more intense and potentially more neurotoxic than for others and informs any future harm reduction decisions. SLC6A4 SS allele explains chronic social anxiety and serotonin depletion susceptibility. MTHFR explains SSRI non-response and the transformative response to 5-MTHF.
Social Environment Redesign (Weeks 1–8)	Protocol 05, Protocol 08, Protocol 12 (Sonic)	Festival/event environments; music associations; social community renegotiation	The sonic protocol is specifically important for MDMA recovery — music is the most powerful MDMA-associated cue for most users. Building a recovery playlist that is not MDMA-associated while managing the MDMA-associated music responsibly is a recovery-specific environmental design task addressed in Protocol 12.
Depression Treatment (Week 1 onwards if indicated)	5-MTHF assessment; psychiatrist if needed; BDNF building (exercise)	Serotonergic depression management without SSRIs where possible; MTHFR assessment before prescription antidepressant trial	The person whose MDMA use was managing depression has not had their depression treated — they have had it chemically masked. 5-MTHF first for MTHFR-likely profiles. Exercise immediately and daily. If clinical depression persists at 4–6 weeks post-cessation with full supplement protocol: psychiatric evaluation.
Connection	Protocol 04,	Sober connection	This is the central recovery project

Architecture (Months 1–6)	Protocol 08, Protocol 22, Section 6 of this guide	building; one-to-one authentic relationship; communal movement; service	of MDMA recovery and the one most underaddressed in standard addiction treatment. Building the connection architecture is not therapy homework — it is the biological reconstruction of the serotonin and oxytocin systems through their natural activation pathways. It must be deliberate, daily, and prioritized.
Identity Work (Months 2–12)	Protocol 20, Protocol 21, C-28	Identity beyond the scene; who am I socially without the chemical permission slip; the authentic self that MDMA was facilitating access to	The identity question in MDMA recovery: 'The person I was on MDMA — the open, connected, loving, present version — is that my real self or was that the drug?' The answer the compendium provides: both. The drug facilitated something real. Recovery is building direct access to it.
Long Game (Months 6–24+)	Protocol 24, Protocol 25, C-28, C-31	Community fully built; serotonin system recovered; connection accessible without chemicals; the social self that MDMA was approximating, now genuinely owned	The person at 12–24 months of MDMA recovery, with rebuilt serotonin system, a genuine community of connection, and the practiced capacity for authentic social presence — reports that the quality of connection is deeper and more real than anything MDMA produced. Because it is built, not borrowed. Because it grows, not depletes. Because it is theirs.

Section 8 — The Medical and Therapeutic Context

Medical Priorities in MDMA Recovery

- CYP2D6 metabolizer status: if MDMA effects were consistently more intense or longer than peers at the same dose, CYP2D6 testing is recommended. This information is relevant for any future medical encounters where CYP2D6-metabolized medications are prescribed.
- Cognitive assessment: if significant memory impairment, word-finding difficulty, or executive function changes persist at 3+ months of abstinence — neuropsychological testing is appropriate. Some heavy MDMA users show measurable cognitive impairment that requires rehabilitation support.
- Psychiatric evaluation: if depression meets clinical criteria at 4–6 weeks post-cessation with full supplement protocol in place — psychiatric evaluation is indicated. The MTHFR assessment should precede SSRI initiation.
- STI assessment: MDMA use in sexual contexts carries STI transmission risk through the same disinhibitory mechanism as chemsex substances. Multi-site STI testing if MDMA was used in sexual contexts without consistent protection.
- Cardiovascular: for users who experienced MDMA-related hyperthermia or cardiac symptoms during use, ECG and cardiovascular assessment is appropriate.

The Therapeutic MDMA Question

THE CENTURION — The Same Molecule — A Different Relationship

MDMA-assisted psychotherapy for PTSD is approved in Australia as of 2023 and is in regulatory review in the United States as of 2026. Clinical trials show significant efficacy for treatment-resistant PTSD. The therapeutic context differs fundamentally from recreational use: lower doses, more infrequent sessions (2–3 over the treatment course), professional therapeutic containment, integration sessions, and explicit focus on the trauma being processed rather than the social experience being sought.

The question for someone with MDMA use disorder: can therapeutic MDMA be used as part of PTSD treatment if PTSD is an underlying driver of the MDMA use?

The Recovery Kernel's position: this is a clinical decision requiring addiction medicine specialist and psychiatrist collaboration, with full disclosure of the MDMA use history to the treating MDMA therapy team. The therapeutic context's controlled dose and frequency may allow therapeutic benefit without triggering use disorder patterns — but this cannot be assumed and requires careful clinical

monitoring. CYP2D6 status is essential information before any therapeutic MDMA session, as poor metabolizers face elevated risk at any dose.

The person most likely to benefit from therapeutic MDMA in this context and most likely to be harmed by it is the same person — the one whose PTSD drove the MDMA use disorder. This paradox requires careful, individualized clinical assessment. It is not a reason to categorically exclude therapeutic MDMA from consideration, nor a reason to pursue it without full clinical oversight.

Medications for Co-Occurring Conditions

- Depression (if clinical at 4–6 weeks): SSRIs or SNRIs with pharmacogenomic guidance. CRITICAL: if you are prescribed an SSRI, do not use MDMA — the combination produces significant serotonin syndrome risk, and SSRIs also dramatically reduce MDMA's subjective effects, potentially driving users to take more and increase neurotoxicity risk.
- Social anxiety disorder: buspirone (non-addictive serotonin 1A partial agonist); SSRIs with CBT. The social anxiety that MDMA was managing requires its own treatment when MDMA is stopped.
- PTSD: trauma-specific treatment (EMDR, CPT, Prolonged Exposure). If therapeutic MDMA is being considered — see above.
- ADHD: if MDMA was also managing ADHD symptoms — non-stimulant ADHD treatment (atomoxetine, guanfacine, viloxazine). Stimulant ADHD treatment requires careful risk-benefit assessment.

A Final Word

THE CENTURION — The Open Heart Was Real. Recovery Is Finding Out It Never Needed the Molecule.

The connection you felt on MDMA was not a hallucination. It was not a lie. It was not a chemical trick your brain was playing on you about feelings that were not real. It was a genuine neurological state — serotonin flooding, oxytocin releasing, the social anxiety that normally mediates every human encounter going quiet — and what you experienced in that state was authentic: the openness to other people, the love for the music and the crowd and the night, the feeling that barriers between human beings are arbitrary and that what is real is the connection underneath them.

That experience was true. It was also borrowed. The molecule that opened the door was simultaneously closing it — depleting the very system whose activation produced the state you were seeking. The door opened and closed at the same time, for years, until the mechanism wore down.

What the Recovery Kernel offers is the map to a door that opens without closing. The serotonin system, rebuilt to its own capacity. The oxytocin system, activated by genuine human presence rather than pharmacology. The social capacity — practiced, imperfect, real — for connection without a chemical intermediary.

The compendium's table is precise: MDMA — Connection, love, openness — Serotonin, social bonding deficit — Authentic connection practices, community.

Three words on each side of the arrow. The work of recovery is the distance between them.

The open heart that MDMA was providing access to has been yours all along. It was never in the molecule. The molecule was the detour. Recovery is the direct route.

— — —

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE